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Connecting via Winsock to Dialog


Logging in to Dialog

Trying 31060000009998...Open

DIALOG INFORMATION SERVICES
PLEASE LOGON:
 ********
ENTER PASSWORD:
 ********
Welcome to DIALOG

Dialog level 05.17.01D

Last logoff:  09jun07 15:08:11
Logon file405 09jun07 15:20:14
* * *
SYSTEM:HOME
Cost is in DialUnits
Menu System II: D2 version 1.8.0 term=ASCII
                     *** DIALOG HOMEBASE(SM) Main Menu ***

 Information:
  1.  Announcements (new files, reloads, etc.)
  2.  Database, Rates, & Command Descriptions
  3.  Help in Choosing Databases for Your Topic
  4.  Customer Services (telephone assistance, training, seminars, etc.)
  5.  Product Descriptions

 Connections:
  6.  DIALOG(R) Document Delivery
  7.  Data Star(R)

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      /H = Help            /L = Logoff          /NOMENU = Command Mode


Enter an option number to view information or to connect to an online
 service.  Enter a BEGIN command plus a file number to search a database
(e.g., B1 for ERIC).
? b 410

       09jun07 15:20:14 User226352 Session D1018.1
            $0.00    0.245 DialUnits FileHomeBase
     $0.00  Estimated cost FileHomeBase
     $0.00  Estimated cost this search
     $0.00  Estimated total session cost   0.245 DialUnits

File 410:Dialog Comm.-of-Interest Newsletters 2007 /Feb
       (c) 2007 Dialog

      Set  Items  Description
      ---  -----  -----------
? set hi ;set hi
HILIGHT set on as ''
HILIGHT set on as ''
? file 1555
>>>        1555 is unauthorized
>>>1 of the specified files is not available
>>>No valid files specified
? b file 155
```

```
>>>"FILE" is not a valid category or service name
       09jun07 15:20:30 User226352 Session D1018.2
            $0.00    0.228 DialUnits File410
     $0.00  Estimated cost File410
     $0.06  TELNET
     $0.06  Estimated cost this search
     $0.06  Estimated total session cost   0.473 DialUnits

File 155:MEDLINE(R) 1950-2007/Jun 08
       (c) format only 2007 Dialog
*File 155: Medline has been reloaded. Please see HELP NEWS 154
for information on 2007 changes.

      Set  Items  Description
      ---  -----  -----------
? e au=vigo-pelfry

Ref   Items  Index-term
E1        1  AU=VIGO-GUEVARA G L
E2       18  AU=VIGO-PELFREY C
E3        0 *AU=VIGO-PELFRY
E4        1  AU=VIGOA L
E5        2  AU=VIGOD SIMONE N
E6        1  AU=VIGODA AYELET
E7        1  AU=VIGODA D S
E8        4  AU=VIGODA ERIC
E9        1  AU=VIGODA M
E10       1  AU=VIGODA MICHAEL
E11       4  AU=VIGODA MICHAEL M
E12       3  AU=VIGODA P S

          Enter P or PAGE for more
? s e2
      S1      18  AU='VIGO-PELFREY C'
? t s1/7/all

 1/7/1
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2007 Dialog. All rts. reserv.

11712752   PMID: 9520072
  Cerebrospinal fluid levels of amyloid precursor protein and amyloid
beta-peptide in Alzheimer's disease and major depression - inverse
correlation with dementia severity.
  Hock C; Golombowski S; Muller-Spahn F; Naser W; Beyreuther K; Monning U;
Schenk D; Vigo-Pelfrey C; Bush A M; Moir R; Tanzi R E; Growdon J H;
Nitsch R M
  Department of Psychiatry, University of Basel, Switzerland.
hock@ubaclu.unibas.ch
  European neurology (SWITZERLAND)      1998, 39    (2)    p111-8, ISSN
0014-3022--Print   Journal Code: 0150760
  Publishing Model Print
  Document type: Clinical Trial; Comparative Study; Controlled Clinical
Trial; Journal Article; Research Support, Non-U.S. Gov't
  Languages: ENGLISH
  Main Citation Owner: NLM
  Record type: MEDLINE; Completed
  Alzheimer's disease (AD) is the most common neurodegenerative disorder
characterized by progressive dementia that ultimately leads to death.
Histopathological hallmarks of AD include brain amyloid deposits and
neurofibrillary tangles. Major depression is a frequent diagnosis in every
gerontopsychiatric clinic that sees patients with both cognitive and
affective disorders. Many depressed patients, in fact, are clinically
characterized by cognitive impairments. Thus, an assay that excludes - or
confirms - probable AD in cognitively impaired patients is desirable. Such
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assays may use protein markers that are derived from such histopathologically relevant molecules as the amyloid precursor protein (APP) and its derivatives including the amyloid beta-peptides (Abeta). To evaluate the differential diagnostic properties of cerebrospinal fluid (CSF) Abeta and secreted soluble ectodomain (APPs), we quantitated CSF levels of these measures in AD patients and compared them to age-matched control patients with major depression. CSF levels of APPs and Abeta were similar in patients with AD or major depression, and the apolipoprotein E genotype had no influence on CSF levels of Abeta in AD patients. Measurement of Abeta peptide using a novel zinc/copper capture ELISA that detects aggregated Abeta peptides as well demonstrated similar levels in AD and major depression. In AD patients, CSF levels of total Abeta (Abeta1-40 plus Abeta1-42) were inversely correlated with a functional measure of dementia severity (NOSGER), suggesting that CSF levels of Abeta decrease with advancing severity of AD. Thus, CSF levels of Abeta are not useful for the differentiation of AD from major depression. However, CSF levels of Abeta reflect the severity of dementia and may be useful as biological markers of the stage of the disease.
Record Date Created: 19980527
Record Date Completed: 19980527

1/7/2
DIALOG(R)File 155:MEDLINE(R)

10968924 PMID: 8780035
Glutamine synthetase-induced enhancement of beta-amyloid peptide A beta (1-40) neurotoxicity accompanied by abrogation of fibril formation and A beta fragmentation.
Aksenov M Y; Aksenova M V; Butterfield D A; Hensley K; Vigo-Pelfrey C; Carney J M
Department of Pharmacology, University of Kentucky, Lexington 40536, USA.
Journal of neurochemistry (UNITED STATES) May 1996, 66 (5) p2050-6, ISSN 0022-3042--Print Journal Code: 2985190R
Contract/Grant No.: AG-09690; AG; NIA; AG-10836; AG; NIA
Publishing Model Print
Document type: Journal Article; Research Support, U.S. Gov't, P.H.S.
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
beta-Amyloid peptide (A beta) is the main constituent in both senile plaques and diffuse deposits in Alzheimer's diseased brains. It was previously shown that synthetic A beta s were able to form free radical species in aqueous solution and cause both oxidative damage to cell proteins and inactivation of key metabolic enzymes. We also previously demonstrated that an interaction of A beta (1-40) with the oxidatively sensitive enzyme glutamine synthetase (GS) resulted in both inactivation of GS and an increase of A beta toxicity to hippocampal cell cultures. In the present study the enhancement of A beta toxicity during interaction with GS was found to be accompanied by abrogation of fibril formation and partial fragmentation of A beta (1-40). HPLC elution profiles demonstrated the production of several peptide fragments. Analysis of the amino acid sequence of the major fragments identified them as the first 15 and the last six amino acids of A beta (1-40). The fragmentation of A beta was inhibited by immunoprecipitation of GS.
Record Date Created: 19970114
Record Date Completed: 19970114

1/7/3
DIALOG(R)File 155:MEDLINE(R)

10850030 PMID: 8626743

Water-soluble Abeta (N-40, N-42) oligomers in normal and Alzheimer disease brains.

Kuo Y M; Emmerling M R; Vigo-Pelfrey C; Kasunic T C; Kirkpatrick J B; Murdoch G H; Ball M J; Roher A E

Sun Health Research Institute, Sun City, Arizona 85372, USA.

Journal of biological chemistry (UNITED STATES) Feb 23 1996, 271 (8) p4077-81, ISSN 0021-9258--Print Journal Code: 2985121R

Contract/Grant No.: 5P50AG-08664; AG; NIA; AG-11925; AG; NIA; P30AG-08017 ; AG; NIA

Publishing Model Print

Document type: Comparative Study; Journal Article; Research Support, Non-U.S. Gov't; Research Support, U.S. Gov't, P.H.S.

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

Ultracentrifugation and graded molecular sieving, as well as a sensitive sandwich enzyme-linked immunosorbent assay were used to isolate and quantitate the amounts of water-soluble oligomers of beta amyloid (Abeta) peptides N-40 and N-42 in cerebral cortex of normal and Alzheimer disease (AD) brains. AD brains contained 6-fold more water-soluble Abeta (wsAbeta) than control brains. The majority of water-soluble peptides in most AD cases was A beta N-42, representing 12 times the amount found in control brains. The wsAbeta was present in the form of monomers and oligomers ranging from less than 10 kDa to greater than 100 kDa. The amount of wsAbeta N-42 in AD brains is about 50 times greater than the level of soluble Abeta N-42 found in the CSF of AD patients. This disparity may be due to the rapid association of wsAbeta N-42 into fibrillar deposits and/or to the integrity of the anatomical barriers which separate the two extracellular spaces. In this paper, we consider soluble any form of Abeta which has not yet polymerized into its insoluble, filamentous form. This includes both the newly synthesized forms of Abeta and those peptides which may be loosely attached to insoluble filaments but which can, nevertheless, still be considered soluble. It has been previously shown that, once it has aggregated into its filamentous form, the Abeta peptides are resistant to disaggregation and degradation by a number of denaturing agents and aqueous buffers containing proteolytic enzymes. Therefore, it is likely that the water-soluble Abeta peptides we quantified are precursors to its insoluble, filamentous form. Consequently, reducing the levels of soluble Abeta in AD brains could have profound effects on AD pathophysiology.

Record Date Created: 19960621

Record Date Completed: 19960621

1/7/4

10724071 PMID: 8577398

Amyloid beta-peptide in cerebrospinal fluid in individuals with the Swedish Alzheimer amyloid precursor protein mutation.

Lannfelt L; Basun H; Vigo-Pelfrey C; Wahlund L O; Winblad B; Lieberburg I; Schenk D

Karolinska Institute, Department of Clinical Neuroscience and Family Medicine, Huddinge University Hospital, Sweden.

Neuroscience letters (IRELAND) Oct 27 1995, 199 (3) p203-6, ISSN 0304-3940--Print Journal Code: 7600130

Publishing Model Print

Document type: Journal Article; Research Support, Non-U.S. Gov't

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

The neuropathological hallmarks of Alzheimer's disease (AD) are amyloid-containing plaques and neurofibrillary tangles. The main constituent of senile plaques is amyloid beta-peptide (A beta) and in recent years, pathogenic mutations in the amyloid precursor protein (APP)

gene have been discovered in some AD families. The APP670/671 mutation, found in a Swedish AD family, has revealed over-production of A beta as one pathogenic mechanism for the development of AD. In the present study we have used an immunoassay to measure A beta levels in cerebrospinal fluid (CSF) from APP670/671 mutation-carriers and non-carriers. A correlation was seen between decrease in A beta levels and duration of disease although no difference was found in levels of A beta between the groups (14.5 +/- 3.3 ng/ml versus 14.9 +/- 2.3 ng/ml).
Record Date Created: 19960313
Record Date Completed: 19960313

1/7/5
DIALOG(R)File 155:MEDLINE(R)

10586422 PMID: 7574461
Reduction of beta-amyloid peptide42 in the cerebrospinal fluid of patients with Alzheimer's disease.
Motter R; Vigo-Pelfrey C; Kholodenko D; Barbour R; Johnson-Wood K; Galasko D; Chang L; Miller B; Clark C; Green R; et al
Athena Neurosciences, Inc, South San Francisco, CA 94080, USA.
Annals of neurology (UNITED STATES) Oct 1995, 38 (4) p643-8, ISSN 0364-5134--Print Journal Code: 7707449
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
In this clinical study the cerebrospinal fluid (CSF) level of a novel form of the beta-amyloid peptide (A beta) extending to position 42 (A beta 42) was determined in patients with Alzheimer's disease (AD) as well as controls. In addition to measurement of CSF A beta 42 levels, total A beta peptides, microtubule-associated protein tau, and apolipoprotein E (ApoE) genotype were also assessed. It is interesting that CSF A beta 42 levels were found to be significantly lower in AD patients relative to controls, whereas total A beta levels were not. A beta 42 has recently been shown to preferentially deposit in the brain tissue of patients with AD, suggesting that diminished clearance may account for its reduction in CSF. As previously reported, tau levels were increased in AD patients; however, neither A beta 42 nor tau levels were apparently influenced by the ApoE genotype.
Record Date Created: 19951109
Record Date Completed: 19951109

1/7/6
DIALOG(R)File 155:MEDLINE(R)

10529057 PMID: 7623120
Chronic elevation of secreted amyloid precursor protein in subcortically lesioned rats, and its exacerbation in aged rats.
Wallace W C; Lieberburg I; Schenk D; Vigo-Pelfrey C; Davis K L; Haroutunian V
Laboratory of Biochemical Genetics, NIMH, Washington, DC, USA.
Journal of neuroscience - the official journal of the Society for Neuroscience (UNITED STATES) Jul 1995, 15 (7 Pt 1) p4896-905, ISSN 0270-6474--Print Journal Code: 8102140
Contract/Grant No.: R01 AG10138; AG; NIA
Publishing Model Print
Document type: Journal Article; Research Support, U.S. Gov't, P.H.S.
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed

Subcortically lesioned rats were used as an animal model of some of the neurochemical and behavioral deficits of Alzheimer's disease (AD) to investigate the in vivo expression and metabolism of amyloid precursor protein (APP). Previously, the rapid and persistent induction of APP was described in cerebral cortices after disruption of its cholinergic, serotonergic, or noradrenergic afferents. In the present study, this induction was found to lead to the elevated secretion of APP into the cerebrospinal fluid of lesioned animals. Lesions of the forebrain cholinergic system in aged rats caused an even greater increase in the CSF levels of secreted APP. Antibodies to the extracellular domain of APP detected the protein whereas antibodies to the cytoplasmic region did not, indicating that the APP present in CSF was of the soluble form. Immunoprecipitation with an A beta sequence-specific antibody followed by immunoblot analysis indicated that a significant portion of secreted APP was of the species that contains at least the first 28 amino acids of the A beta sequence (APP gamma or APPA beta). By contrast, very low levels of A beta peptide were detected in CSF. The secretion was accompanied by an elevation of cellular C-terminal fragments of the APP in the lesioned cortex. Consistent with our previous results, this increased APP secretion was caused by lesions of subcortical cholinergic and serotonergic systems. The postlesion time course of APP secretion showed an initial reduction of APP (1 hr postlesion) in CSF followed by an eventual twofold elevation 1-6 weeks later. These results indicate that the induction of APP in response to loss of subcortical innervation leads to elevated secretion of a soluble form of cortically derived APP that contains significant portions of the A beta sequence.

Record Date Created: 19950828
Record Date Completed: 19950828

1/7/7
DIALOG(R)File 155:MEDLINE(R)

10434980 PMID: 7723971

Elevation of microtubule-associated protein tau in the cerebrospinal fluid of patients with Alzheimer's disease.

Vigo-Pelfrey C; Seubert P; Barbour R; Blomquist C; Lee M; Lee D; Coria F; Chang L; Miller B; Lieberburg I; et al
Athena Neurosciences, Inc, South San Francisco, CA 94080, USA.

Neurology (UNITED STATES) Apr 1995, 45 (4) p788-93, ISSN 0028-3878 --Print Journal Code: 0401060
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed

Currently, there is no biochemical marker clinically available to test for the presence of Alzheimer's disease (AD). Recent studies suggest that the core component of AD-associated neurofibrillary tangles (NFTs), the microtubule-associated protein tau, might be present in CSF. This study focuses on establishing both the presence of tau in CSF and its potential utility in the diagnosis of AD. We obtained CSF from 181 individuals; 71 of these were diagnosed as having probable AD by NINCDS-ADRDA criteria. The remaining 110 individuals were divided into three groups: (1) age-matched demented non-AD patients (n = 25), (2) neurologic controls (n = 59), and (3) other controls (n = 26). We developed a sensitive enzyme-linked immunosorbent tau assay using monoclonal antibodies prepared against recombinant human tau. We confirmed specificity of the antibodies by a combination of immunoprecipitation and immunoblot results. By this assay we measured that the AD population has a mean level of tau 50% greater than the non-AD dementia patients. Comparing AD patients with all other groups, the difference in tau levels as analyzed by one-way ANOVA is highly statistically significant ($p < 0.001$). Postmortem analysis of two AD patients with high levels of CSF tau revealed a high density of NFTs in the

hippocampus. There was no significant correlation between tau and age in the non-AD groups. This study suggests that CSF tau is elevated in AD and might be a useful aid in antemortem diagnosis.
Record Date Created: 19950523
Record Date Completed: 19950523

1/7/8
DIALOG(R)File 155:MEDLINE(R)

10429604 PMID: 7717688
Cerebrospinal fluid levels of amyloid beta-protein in Alzheimer's disease: inverse correlation with severity of dementia and effect of apolipoprotein E genotype.
Nitsch R M; Rebeck G W; Deng M; Richardson U I; Tennis M; Schenk D B; Vigo-Pelfrey C; Lieberburg I; Wurtman R J; Hyman B T; et al
Department of Neurology, Massachusetts General Hospital, Boston, USA.
Annals of neurology (UNITED STATES) Apr 1995, 37 (4) p512-8, ISSN 0364-5134--Print Journal Code: 7707449
Contract/Grant No.: 2P50 AG-01534; AG; NIA; NIMH 28783; MH; NIMH; RR-01066; RR; NCRR
Publishing Model Print
Document type: Journal Article; Research Support, Non-U.S. Gov't; Research Support, U.S. Gov't, P.H.S.
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Alzheimer's disease (AD) is characterized by formation in brain of neurofibrillary tangles and of amyloid deposits. The major protein component of the former is tau, while the latter are composed of amyloid beta-peptides (A beta), which are derived by proteolytic cleavage of the amyloid beta-protein precursor (APP). Both tau and various secretory APP derivatives including A beta and APPS are present in human cerebrospinal fluid (CSF). To investigate whether clinical signs of AD are paralleled by changes in CSF levels of these proteins, we correlated quantitative measures of dementia severity with CSF concentrations of A beta, of APPS, and of tau. We found that levels of A beta in CSF of AD patients were inversely correlated both to cognitive and to functional measures of dementia severity. In contrast, levels of APPS and of tau did not correlate with dementia severity. Apolipoprotein E (apoE) genotype did not influence CSF levels of A beta, APPS, or tau, which were similar among AD patients with Apo E epsilon 3/3, epsilon 3/4, and epsilon 4/4 alleles. These data indicate that CSF levels of A beta decrease with advancing severity of dementia in AD and suggest that they are independent of a patient's Apo E genotype.
Record Date Created: 19950515
Record Date Completed: 19950515

1/7/9
DIALOG(R)File 155:MEDLINE(R)

10250460 PMID: 7991571
Excessive production of amyloid beta-protein by peripheral cells of symptomatic and presymptomatic patients carrying the Swedish familial Alzheimer disease mutation.
Citron M; Vigo-Pelfrey C; Teplow D B; Miller C; Schenk D; Johnston J; Winblad B; Venizelos N; Lannfelt L; Selkoe D J
Department of Neurology, Harvard Medical School, Boston, MA.
Proceedings of the National Academy of Sciences of the United States of America (UNITED STATES) Dec 6 1994, 91 (25) p11993-7, ISSN 0027-8424 --Print Journal Code: 7505876
Contract/Grant No.: AG06173; AG; NIA; AG07911; AG; NIA

Publishing Model Print
Document type: Journal Article; Research Support, Non-U.S. Gov't; Research Support, U.S. Gov't, P.H.S.
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed

The 39- to 43-amino acid amyloid beta-protein (A beta), which is progressively deposited in cerebral plaques and blood vessels in Alzheimer disease (AD), is secreted by cultured human cells during normal metabolism. In studies of cell lines transfected with beta-amyloid precursor protein (beta APP) cDNAs, the beta APP mutation K670N/M671L found in a Swedish familial AD (FAD) pedigree has previously been shown to cause a marked augmentation of A beta secretion. Here, we have conducted blinded analyses of beta APP metabolism in primary skin fibroblasts from affected members of the Swedish FAD pedigree and their unaffected siblings or spouses. These fibroblasts continuously secrete a homogenous population of A beta molecules starting at Asp-1 (D672 of beta APP). We found a consistent and significant approximately 3-fold elevation of A beta release from all biopsied skin fibroblasts bearing the FAD mutation. No significant alterations of other metabolic derivatives of beta APP were detected. The elevated A beta levels were found in cells from both patients with clinical AD and presymptomatic subjects. Thus, A beta overproduction in this FAD pedigree is not a secondary event but is consistent with a causal role in the development of the disease. Increased A beta secretion can begin many years prior to onset of symptoms, even in peripheral tissues, indicating that it does not require preexisting neural abnormalities.

Record Date Created: 19950111
Record Date Completed: 19950111

1/7/10
DIALOG(R)File 155:MEDLINE(R)

10214621 PMID: 7957938

Increased beta-amyloid release and levels of amyloid precursor protein (APP) in fibroblast cell lines from family members with the Swedish Alzheimer's disease APP670/671 mutation.

Johnston J A; Cowburn R F; Norgren S; Wiehager B; Venizelos N; Winblad B; Vigo-Pelfrey C; Schenk D; Lannfelt L; O'Neill C
Department of Geriatric Medicine, Karolinska Institute, Huddinge, Sweden.

FEBS letters (NETHERLANDS) Nov 14 1994, 354 (3) p274-8, ISSN 0014-5793--Print Journal Code: 0155157
Publishing Model Print
Document type: Journal Article; Research Support, Non-U.S. Gov't
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed

Cell lines transfected with the Swedish Alzheimer's disease amyloid precursor protein APP670/671 mutation release significantly more beta-amyloid than wild-type cells. Citron et al. [Proc. Natl. Acad. Sci. USA (1994) in press] have recently shown that fibroblasts carrying the APP670/671 mutation also release more beta-amyloid than control cells [1]. The present study confirms a ca. threefold increase in beta-amyloid release from mutation-bearing fibroblasts. APP mRNA levels did not differ between mutation-bearing and control cells, although mutation-bearing fibroblasts contained significantly more APP751/770 than controls. Mild stress decreased beta-amyloid secretion and increased APP751/770 levels in all cell lines. In conclusion, the proportion of APP committed to amyloidogenic processing is increased in fibroblasts from family members with the APP670/671 mutation, and this mutation may also compromise the APP stress response.

Record Date Created: 19941220
Record Date Completed: 19941220

1/7/11
DIALOG(R)File 155:MEDLINE(R)

09846708 PMID: 8242833
Quality control of liposomal lipids with special emphasis on peroxidation of phospholipids and cholesterol.
Lang J K; Vigo-Pelfrey C
Liposome Technology, Inc., Menlo Park, CA 94025.
Chemistry and physics of lipids (IRELAND) Sep 1993, 64 (1-3) p19-29, ISSN 0009-3084--Print Journal Code: 0067206
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
The usefulness of various assays for the determination of phospholipid and cholesterol peroxidation in liposome formulations was studied on model liposomes prepared as small unilamellar vesicles (SUV) and multilamellar vesicles (MLV) from either native egg phosphatidylcholine (EPC), partially hydrogenated egg phosphatidylcholine (PHEPC) or fully hydrogenated egg phosphatidylcholine (HEPC) and cholesterol in 65/35 molar ratio at a total lipid concentration of 10 mumol/ml in phosphate buffered saline pH 7.2. Liposomes were incubated at 50 degrees C for a total of 3 months. Fatty acid and cholesterol peroxidation were monitored after 1, 2 and 3 months by quantitative measurement of fatty acids and cholesterol and as well as peroxidation products. Fatty acid peroxidation products malondialdehyde, lipidhydroperoxides, conjugated dienes, conjugated trienes were poor predictors of actual fatty acid loss. Among the cholesterol peroxidation products 7-hydroxy-cholesterols, 7-keto-cholesterol and 4-cholesten-3-one were measured quantitatively. Only the formation of 7-keto-cholesterol correlated well with cholesterol disappearance.
Record Date Created: 19931229
Record Date Completed: 19931229

1/7/12
DIALOG(R)File 155:MEDLINE(R)

09830752 PMID: 8229004
Characterization of beta-amyloid peptide from human cerebrospinal fluid.
Vigo-Pelfrey C; Lee D; Keim P; Lieberburg I; Schenk D B
Athena Neurosciences, Inc. South San Francisco, CA 94080.
Journal of neurochemistry (UNITED STATES) Nov 1993, 61 (5) p1965-8, ISSN 0022-3042--Print Journal Code: 2985190R
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
beta-Amyloid peptide (A beta) is one of the main components of senile plaques in the brain tissue of Alzheimer's disease (AD) patients. A beta is proteolytically cleaved from the amyloid precursor protein (APP), an integral membrane protein possessing a large extracellular N-terminal domain followed by a single membrane-spanning region and a short cytoplasmic C-terminal tail. A beta has been isolated from senile plaques and cerebral vascular tissue of AD brain and characterized as a heterogeneous peptide containing 28-43 amino acids whose sequence begins in the extracellular domain of APP and extends into the putative transmembrane sequence. It has long been speculated that A beta may also be present in body fluids, such as CSF, that contact neuritic plaques. Recently using a specific enzyme-linked immunosorbent assay we were able to quantify one form of A beta in CSF. In this report, using one of these antibodies

covalently bound as an affinity matrix, multiple complex forms of A beta have been isolated and characterized from CSF derived from patients with either meningitis or other neurological disorders. Amino acid sequencing reveals A beta species with N-termini of Asp1, Glu3, His6, Glu11, and Val12, although on a molar basis, Asp1 represents the predominant aminoterminus. Laser desorption mass spectrometry confirmed the presence in CSF of A beta species containing 27, 28, 30, 34, 35, 40, 42, and 43 amino acids, all beginning at Asp1; two stable trimers, (Asp1-Met35)3 and (His6-Ala42)3; and one stable dimer containing (Asp1-Val40)2.(ABSTRACT TRUNCATED AT 250 WORDS)
Record Date Created: 19931129
Record Date Completed: 19931129

1/7/13
DIALOG(R)File 155:MEDLINE(R)

09752994 PMID: 8353995
Comparison of a fluorometric test with the standard ELISA assay for the detection of anticardiolipin antibodies.
Pierangeli S S; Vigo-Pelfrey C; Harris E N
Clinical and experimental rheumatology (ITALY) May-Jun 1993, 11 (3) p349-50, ISSN 0392-856X--Print Journal Code: 8308521
Publishing Model Print
Document type: Comparative Study; Letter
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Record Date Created: 19930923
Record Date Completed: 19930923

1/7/14
DIALOG(R)File 155:MEDLINE(R)

09487320 PMID: 1465129
Mutation of the beta-amyloid precursor protein in familial Alzheimer's disease increases beta-protein production.
Citron M; Oltersdorf T; Haass C; McConlogue L; Hung A Y; Seubert P; Vigo-Pelfrey C; Lieberburg I; Selkoe D J
Department of Neurology, Harvard Medical School, Boston, Massachusetts.
Nature (ENGLAND) Dec 17 1992, 360 (6405) p672-4, ISSN 0028-0836--Print Journal Code: 0410462
Publishing Model Print
Document type: Journal Article; Research Support, Non-U.S. Gov't; Research Support, U.S. Gov't, P.H.S.
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Progressive cerebral deposition of the 39-43-amino-acid amyloid beta-protein (A beta) is an invariant feature of Alzheimer's disease which precedes symptoms of dementia by years or decades. The only specific molecular defects that cause Alzheimer's disease which have been identified so far are missense mutations in the gene encoding the beta-amyloid precursor protein (beta-APP) in certain families with an autosomal dominant form of the disease (familial Alzheimer's disease, or FAD). These mutations are located within or immediately flanking the A beta region of beta-APP, but the mechanism by which they cause the pathological phenotype of early and accelerated A beta deposition is unknown. Here we report that cultured cells which express a beta-APP complementary DNA bearing a double mutation (Lys to Asn at residue 595 plus Met to Leu at position 596) found in a Swedish FAD family produce approximately 6-8-fold more A beta than cells expressing normal beta-APP. The Met 596 to Leu mutation is principally

responsible for the increase. These data establish a direct link between a FAD genotype and the clinicopathological phenotype. Further, they confirm the relevance of the continuous A beta production by cultured cells for elucidating the fundamental mechanism of Alzheimer's disease.
Record Date Created: 19930121
Record Date Completed: 19930121

1/7/15
DIALOG(R)File 155:MEDLINE(R)

09413912 PMID: 1406936
Isolation and quantification of soluble Alzheimer's beta-peptide from biological fluids.
Seubert P; Vigo-Pelfrey C; Esch F; Lee M; Dovey H; Davis D; Sinha S ; Schlossmacher M; Whaley J; Swindlehurst C; et al
Athena Neurosciences Inc., South San Francisco, California 94080.
Nature (ENGLAND) Sep 24 1992, 359 (6393) p325-7, ISSN 0028-0836--Print Journal Code: 0410462
Publishing Model Print; Comment in Nature. 1992 Sep 24;359(6393) 268-9; Comment in PMID 1406927
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Cerebral deposition of the beta-amyloid peptide (A beta) is an invariant feature of Alzheimer's disease. Since the original isolation and characterization of A beta (ref. 1) and the subsequent cloning of its precursor protein, no direct evidence for the actual production of discrete A beta has been reported. Here we investigate whether A beta is present in human biological fluids using antibodies specific for an epitope within A beta that spans the site of normal constitutive cleavage. These antibodies were used to construct a sandwich-type enzyme-linked immunosorbent assay that detects A beta in cerebrospinal fluid, plasma and conditioned medium of human mixed-brain cells grown in vitro (see also ref. 14). By affinity chromatography, we have purified and sequenced A beta and a novel A beta fragment from human cerebrospinal fluid and conditioned medium of human mixed-brain cell cultures. These findings demonstrate that A beta is produced and released both in vivo and in vitro. These observations offer new opportunities for developing diagnostic tests for Alzheimer's disease and therapeutic strategies aimed at reducing the cerebral deposition of A beta.
Record Date Created: 19921028
Record Date Completed: 19921028

1/7/16
DIALOG(R)File 155:MEDLINE(R)

09413911 PMID: 1383826
Amyloid beta-peptide is produced by cultured cells during normal metabolism.
Haass C; Schlossmacher M G; Hung A Y; Vigo-Pelfrey C; Mellon A; Ostaszewski B L; Lieberburg I; Koo E H; Schenk D; Teplow D B; et al
Department of Neurology and Program in Neuroscience, Harvard Medical School, Boston, Massachusetts 02155.
Nature (ENGLAND) Sep 24 1992, 359 (6393) p322-5, ISSN 0028-0836--Print Journal Code: 0410462
Publishing Model Print; Comment in Nature. 1992 Sep 24;359(6393) 268-9; Comment in PMID 1406927
Document type: Journal Article; Research Support, Non-U.S. Gov't; Research Support, U.S. Gov't, P.H.S.
Languages: ENGLISH

Main Citation Owner: NLM
Record type: MEDLINE; Completed

Alzheimer's disease is characterized by the extracellular deposition in the brain and its blood vessels of insoluble aggregates of the amyloid beta-peptide (A beta), a fragment, of about 40 amino acids in length, of the integral membrane protein beta-amyloid precursor protein (beta-APP). The mechanism of extracellular accumulation of A beta in brain is unknown and no simple in vitro or in vivo model systems that produce extracellular A beta have been described. We report here the unexpected identification of the 4K (M(r) 4,000) A beta and a truncated form of A beta (approximately 3K) in media from cultures of primary cells and untransfected and beta-APP-transfected cell lines grown under normal conditions. These peptides were immunoprecipitated readily from culture medium by A beta-specific antibodies and their identities confirmed by sequencing. The concept that pathological processes are responsible for the production of A beta must not be reassessed in light of the observation that A beta is produced in soluble form in vitro and in vivo during normal cellular metabolism. Further, these findings provide the basis for using simple cell culture systems to identify drugs that block the formation or release of A beta, the primary protein constituent of the senile plaques of Alzheimer's disease.

Record Date Created: 19921028
Record Date Completed: 19921028

1/7/17
DIALOG(R)File 155:MEDLINE(R)

09198102 PMID: 1537905

Serodiagnosis of Lyme borreliosis by western immunoblot: reactivity of various significant antibodies against Borrelia burgdorferi.

Ma B; Christen B; Leung D; Vigo-Pelfrey C
Whittaker Bioproducts, Inc., Walkersville, Maryland 21793-0127.

Journal of clinical microbiology (UNITED STATES) Feb 1992, 30 (2) p370-6, ISSN 0095-1137--Print Journal Code: 7505564
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed

The significance of various antibodies against Borrelia burgdorferi was studied by Western blot (immunoblot) by using 578 human serum samples. The proteins regularly detected by using samples from patients with Lyme borreliosis were those with bands with molecular masses of 94, 83, 75, 66, 60, 55, 46, 41, 39, 34, 31, 29, 22, and 17 kDa. The detectable frequencies of most of these proteins appeared to be significantly different between the sera from patients with Lyme borreliosis and those from normal control individuals as well as from the group with syphilis. The 39-kDa protein band recognized by polyvalent antibody was found to be the most significant marker for Lyme borreliosis. Furthermore, an anti-39-kDa immunoglobulin M response was detected in the samples from patients with early-stage Lyme borreliosis. Results from the use of monoclonal antibodies and patient sera revealed that the 39- and 41-kDa proteins may be structurally related but are immunologically distinct antigens. The significance of antibody reactivities to the 41-, 94-, 22-, 31-, and 34-kDa protein bands is also discussed.

Record Date Created: 19920330
Record Date Completed: 19920330

1/7/18
DIALOG(R)File 155:MEDLINE(R)

08510216 PMID: 2354545
Liposomes composed of partially hydrogenated egg phosphatidylcholines: fatty acid composition, thermal phase behavior and oxidative stability.
Lang J; Vigo-Pelfrey C; Martin F
Liposome Technology, Inc., Menlo Park, CA 94025.
Chemistry and physics of lipids (NETHERLANDS) Mar 1990, 53 (1) p91-101, ISSN 0009-3084--Print Journal Code: 0067206
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Partially hydrogenated egg phosphatidylcholines (PHEPC) represent a new class of raw materials for liposome-based drug products. PHEPC were manufactured from native egg phosphatidylcholine (EPC) to iodine values (IV) 40, 30, 20, 10, and 1. Hydrogenation resulted in a complete loss of arachidonic acid (20:4) and docosahexaenoic acid (22:6) in IV 40 EPC and a progressive conversion of linoleic (18:2) and oleic acid (18:1) to stearic acid (18:0) at higher degrees of hydrogenation (IV 30, 20, 10, 1). Hydrogenation lead to formation of trans-fatty acid isomers maximally 18.5 mol% in IV 20 EPC. Liposomes made from IV 20, IV 10 and IV 1 EPC had marked phase transitions between 20 and 60 degrees C. PHEPC showed increased resistance to oxidation as measured by oxygen uptake during 2,2'-azobis-(2-amidinopropane) hydrochloride (AAPH) initiated accelerated oxidation. Various applications of these new materials in the manufacture of liposomes and liposome based drug products are discussed.
Record Date Created: 19900720
Record Date Completed: 19900720
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